-- A R S E N I C A N D I T S C O M P O U N D S --

(Arsenic) (Arsenic Trioxide) (Arsenic Chloride)
(Copper Arsenite) (Copper Aceto Arsenite)
(Arsine) (Cacodylic Acid)
(Ethyl Arsine) (Cacodyl)

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ARSENIC AND ITS COMPOUNDS

Arsenic and its compounds have been known from ancient times and it is found in nature in both the free and combined states. Arsenic was extensively used as an agent for secret poisoning during the latter part of the 17th century, one Italian woman being credited with the death of 600 people.

The danger of arsenic poisoning from wall paper was pointed out by Gmelin in 1839. It has since been shown that arsine and its derivatives may be produced by the action of certain molds on wall papers colored with arsenic bearing pigments. Poisoning may also occur from the consumption of beverages such as beer and wine from which spray residues have not been removed.

Certain important industrial metals, particularly zinc, often contain significant traces of arsenic. In the presence of acids such metals give off hydrogen which converts the arsenic present into arsine. Such incidents, all too common in industry, frequently result in death of the workers exposed to the arsine gas. Accidental poisoning by arsine gas undoubtedly constitutes the most serious industrial hazard so far as arsenic and its compounds are concerned.

GENERAL INFORMATION

CHEMICAL FORMULA AND SYNONYMS:

(Arsenic) As_4.

(Arsenic chloride) $AsCl_3$, arsenic trichloride, butter of arsenic, caustic oil of arsenic.

(Arsenic trioxide) As_2O_3, arsenious acid, arsenious oxide, arsenious anhydride, white arsenic.

(Arsine) AsH_3, arsenic trihydrate, arseniuretted hydrogen, arsenious hydride, arsonia, hydrogen arsenide.

(Ethyl arsine) $C_2H_5\text{-}AsH_2$.

(Copper arsenite) $CuHAsO_3$, arsenite of copper, copper orthoarsenite; cupric arsenite, Sheele's green.

(Copper aceto arsenite) $(CuOAs_2O_3)_3\text{-}Cu(C_2H_3O_2)_2$, acetic arsenite of copper; cupric aceto arsenite; emerald green; emperor green; imperial green; Kaiser green; King's green; meadow green; mitis green; moss green; new green; Paris green; parrot green; patent green; Schweinfurth green; Vienna green.

(Cacodylic acid) $(CH_3)_2AsO\text{-}OH$, dimethyl arsenic acid; kakodylic acid.

(Cacodyl) $[(CH_3)_2As]_2$, alkarsin.

PROPERTIES:

(Arsenic) Silvery brittle crystalline metal turning black in air. Sometimes found native. Sp. gr. 5.727 at 14°C.; m.p. 814°C. at 36 atm.; subl. 615°C.; hardness 3.5. Insoluble in water; soluble in nitric acid.

(Arsenic chloride) Colorless oily liquid. Sp. gr. 2.163; m.p.-18°C.; b.p. 130.2°C.; wt. per liter of vapor, 7.55 gr.

(Arsenic trioxide) White amorphous, odorless, tasteless powder; poisonous! Sp. gr. 3.865; m.p. 200°C. Soluble in water, alcohol, acids and alkalis. Wt. per liter of vapor 8.23 gr.

(Arsine) Colorless gas; extremely poisonous! Sp. gr. 2.695; m.p.-113.5°C.; b.p. -55°C., decomposes at 230°C.; wt. per liter, 3.24 gr.

(Ethyl arsine) Colorless liquid. Sp. gr. 1.217 at 22°C.; b.p. 36°C. Slightly soluble in water.

(Copper arsenite) Fine, light green powder; poisonous! M.P.: decomposes. Soluble in acids; insoluble in water.

(Copper aceto arsenite) Emerald green powder; poisonous! Soluble in acids; insoluble in alcohol and water.

(Cacodylic acid) Colorless, odorless, deliquescent crystals; poisonous! M.P. 200°C. Soluble in water and alcohol.

(Cacodyl) Colorless oil. Sp. gr. more than 1; m.p. -6°C.; b.p. about 170°C. Very slightly soluble in water; soluble in alcohol and ether. Compounds are noted for their vile odor and poisonous properties.

OCCURRENCE:

(Arsenic) Arsenopyrite, mimetite, nicolite, orpiment, realgar, scorodite, smaltite, sperrylite.

(Arsine) By the action of sulfuric acid on metallic zinc contaminated with arsenic compounds.

PREPARATION:

(Arsenic) Arsenic ores are roasted and the product recovered by sublimation or distillation. The arsenic oxide thus obtained is reduced.

(Arsenic chloride) (a) By action of chlorine on arsenic. (b) By distillation of arsenic with mercuric chloride. (c) By distillation of arsenious oxide with strong hydrochloric acid. (d) By heating dry arsenious oxide with sulfur chloride at 100° to 125°C.

(Arsenic trioxide) By roasting arsenical pyrites (mispickel) and recovery of the arsenic trioxide by sublimation.

(Arsine) Action of nascent hydrogen on arsenic.

(Ethyl arsine) Reduction of ethyl dichloroarsine.

(Copper arsenite) By the interaction of copper sulfate and sodium arsenite.

(Copper aceto arsenite) By boiling copper basic acetate with arsenic trioxide.

(Cacodylic acid) By distilling a mixture of arsenic trioxide and potassium acetate.

IMPORTANT COMPOUNDS:

(Arsenic) Arsenous acid; arsenic bisulfide; arsenic, black; arsenic bromide; arsenic chloride; arsenic iodide; arsenic pentasulfide; arsenic pentoxide; arsenic trioxide; arsenopyrite; arsine; arsphenamine; diphenyl chloroarsine.

USES:

(Arsenic) Medicine (mercury amalgam); arsenic salts; metallurgy; glass.

(Arsenic chloride) Manufacture of carbon tetrachloride from carbon disulfide (catalyst), pharmaceuticals (arsenated albumins, drug), ceramics (luster finishes).

(Arsenic trioxide) Manufacture of pigments, glass, shot and bullets; insecticides, rat poison, cattle dip, weed killer, hide preservative; medicine (violent irritant); manufacture of other arsenic compounds, ceramic enamels, aniline colors, mixed with soda ash for boiler compound, textile mordant, sterilizing agent in water purification.

(Arsine) Organic synthesis.

(Copper arsenite) Pigment (paints, wall paper, calico printing); insecticide.

(Copper aceto arsenite) Pigment, insecticide, wood preservative preparations.

(Cacodylic acid) Synthesis of dyes, drugs and perfumes.

INDUSTRIAL HEALTH ASPECTS

MODES OF ENTRANCE:

(Arsenic) Inhalation or ingestion.

(Arsenic trioxide) Inhalation or ingestion.

(Arsenic chloride) Inhalation or ingestion.

(Copper arsenite) Inhalation or ingestion.

(Copper aceto arsenite) Inhalation or ingestion.

(Arsine) Inhalation.

(Ethyl arsine) Inhalation.

(Cacodylic acid) Inhalation or ingestion.

(Cacodyl) Inhalation or ingestion.

SYMPTOMS OF INDUSTRIAL POISONING:

Pure arsenic does not appear to be toxic, but its compounds are exceedingly poisonous. Arsenic containing its oxides should be considered harmful; the sulfides of arsenic, if pure, are insoluble and thus not toxic, but commercial products always contain amounts of arsenic acid, the fatal dose of which varies between 1 and 12 cg. according to Lehmann.

Industrial poisoning by arsenic is generally chronic and is usually brought about by the inhalation of arsenical dusts. Acute conditions are at times seen due to release of arsine (arseniuretted hydrogen) in industrial processes.

The clinical signs of arsenic poisoning are generally grouped around the toxic effects on the digestive system, the liver, kidneys, circulatory system and heart. In acute attacks, symptoms of the gastric and renal system predominate; in chronic attacks, symptoms referable to the nervous system and a local action on the skin.

A very acute form arising from massive ingestion of arsenious acid is described, but it is exceptional in industry. Gastric symptoms are severe; there is abdominal colic, nausea, incessant vomiting, and a feeling of dryness in the mouth and thirst. A few hours later, diarrhea, suppression of urine and anuria supervenes, and death advances rapidly, the patient being pale and cyanosed, the appearance resembling cholera.

The acute form is more frequent and is seen in cases of suicide, accidents and occasionally industrially. The dose is high and death usually supervenes in a few days totwo weeks, but because of early vomiting the

poison may be largely rejected and a cure follow. Nausea, vomiting and diarrhea are frequent but usually cease in 24 to 48 hours. Of importance is the fact that the patient then shows a frequent remission of symptoms, improvement generally occurring with a feeling of well-being. However, in a day or two the general conditions again becomes worse with various skin eruptions (scarlatiniform, morbiliform, urticarial purpuric, etc.), labored breathing, cyanosis, cold extremities, small weak pulse, the urine is loaded with albumen and death follows in a state of syncope. If a cure takes place, convalescence is long with frequent gastro-intestinal trouble, chronic nephritis, and often paralysis; but fatal syncope may occur even after a long period of time.

The chronic form is the most frequent seen industrially and follows daily doses of the poison insufficient to cause death but producing organic changes. The first signs are digestive troubles, salivation, nausea, vomiting, abdominal colic, diarrhea, pains in the bones, toxic polyneuritis of the small muscles of the hands and feet, signs of paralysis in lower limbs with numbness, tingling or itching, and anesthesia. If the poisoning continues, various skin eruptions occur, palmo-planter keratosis, and bronzing of the skin (melanodorma). The nails show trophic changes and the hair may fall out. Signs of a cold with laryngitis, hoarseness and cough may be present. Cerebral symptoms of a toxic delirium evidenced by a mental confusion, Korsakoff syndrome, etc., may occur. Motor affections are rarely seen industrially; they consist of peripheral paralysis starting in the lower limbs and progressing upward to involve the upper limbs in about one-half of the cases with abolition of reflexes and frequently anesthesia.

Local action occurs at the point where caustic derivatives of arsenic frequently contact as ulceration of the finger ends, corners of the mouth, or genital organs. Painless ulceration of the septum of the nose is frequent due to the arsenious acid which forms when arsenic dust contacts the moist mucous membranes.

Authorities differ as to the role of arsenic in the production of cancer. The researches of certain experts (Bayet, Slosse, etc.) tend to show that arsenic has a cancerigenetic action. This view is not accepted by some German and English authorities. The English view is that arsenic present in tar does not play a role in the production of epitheliomatous ulceration among tar and pitch workers.

Delepine showed that arsenic can be found in the hair and urine of persons exposed to toxic vapors. It has been found by chemical analysis in various organs as the liver, intestines, kidneys, brain, etc.

In considering the action of arsenic, account must be taken of such things as intolerance or acclimatization, age, state of digestive tract, amount of fatty food in the diet, etc.

According to Hamilton, the elimination of arsenic is very slow.

(Arsenic Trioxide) Historically, it is the most important of the poisons used for criminal purposes. It is a powerful poison and it is stated that two grains may be fatal. It produces inflammation of the stomach and

bowels, violent purging and vomiting, profound nervous collapse, hemolysis with jaundice, urine scanty, bloody or suppressed. If death does not occur, there follows sensory nervous disturbances, neuralgic pains, paresthesias; later, paralysis, loss of hair, deformities of the nails, all sorts of skin lesions, and laryngo-bronchial catarrh. Perforation of the nasal septum has been reported in men handling arsenic trioxide.

(Arsenic chloride) There may be symptoms of intense irritation, itching or ulceration of the skin and mucous membranes, multiple neuritis with sensory and motor disturbances; sweating of palms and soles, and loss of hair, etc. Delepine showed experimentally that arsenic trichloride applied to the skin could set up poisoning.

(Copper arsenite) Symptoms of arsenic poisoning.

(Copper aceto arsenite) Symptoms of arsenic poisoning.

(Arsine) Arsine may be formed as a by-product in industry when nascent hydrogen present reacts with traces of arsenic in metals or acids, etc. It has an unpleasant garlic smell and is extremely toxic, being a powerful hemolytic poison. It is stated that the early symptoms are those of anoxemia; the later symptoms being largely those due to the effort of the body to excrete the debris of red cells which clog the liver and kidneys. A period of time is required after the inhalation of the gas before the products of hemolysis become evident and symptoms result. This period of time varies with the doses from 6 to 36 hours, according to Legge.

It is stated that an atmosphere of 1 part arsine to 4,000 air is rapidly fatal. According to Zangger, a dilution of 1 to 100,000 is poisonous after an absorption of long duration. Dubitzki found arsine to be 10 to 20 times more poisonous than carbon monoxide.

In slight poisoning, there is simply lassitude, headache, malaise, slight dyspnea, occasionally fainting, weak, quick pulse, fall in blood pressure, and sometimes a yellowish coloration of the skin with presence of slight amounts of arsenic in the urine (5 cases have been reported by Wignall). In very slight cases, a cure is effected in a few days after elimination of the exposure.

In average or moderate poisoning, there occurs about 4 to 9 hours after inhalation, lassitude, vertigo, shivering, nausea, vomiting, diarrhea, syncope, oppression and pain in the gastric renal or hepatic regions. Hemoglobinuria appears in 4 to 6 hours; the urine is reddish brown, and for several days contains blood and bile pigments. Jaundice appears in 2 to 3 days. Convalescence is fairly long, albuminuria persists, the blood and bile pigments disappear from the urine and strength is slowly regained.

In serious cases, the onset occurs with a feeling of increasing malaise and weakness, shivering, fatigue, headache, nausea, vomiting, paleness, garlic smell on the breath, pain in hepatic and epigastric regions, and kidney tenderness, weakness, somnolence, semi-unconsciousness, giddiness with restlessness and insomnia and dry throat and thirst. There is anoxemia,

cyanosis, dyspnoea, increased respiration, and syncope may result. After a few hours (about 8-12), evidences of hemolysis appears, the urine is bloody and contains albumin; it may decrease in quantity, oliguria developing into anuria accompanied by pains in kidney region, and convulsions. The pupils may be contracted and react slowly, the conjunctiva may be hemorrhagic or congested. The reflexes may be exaggerated, muscular and nervous pains may occur in the upper or lower limbs. Severe jaundice of the skin and mucous membranes may occur about the second day. On palpation, the liver, gallbladder and spleen may be noted enlarged and pain elicited in the kidney region. The bile production is increased several fold. If recovery results, there is slow convalescence with enemia, weakness, and often a toxic polyneuritis.

(Ethyl arsine) When high concentrations are inhaled, death results almost immediately. The danger arising from arsenical wall papers is stated to be due to liberation of diethyl arsine from decomposing arsenic in the colors by the action of a mold, penicillum glaucum developing in the starch paste on damp walls.

(Cacodylic acid) Action similar to that of ethyl arsine.

(Cacodyl) Action similar to that of ethyl arsine.

INDUSTRIES AND OCCUPATIONS

INDUSTRIES: Ohio Industries using arsenic as indicated in the Ohio Industrial Hygiene Survey are listed as follows:

Chemicals
Electric fixtures
Explosives, ammunition, and fireworks
Foods
Glass factories
Metal furniture

OCCUPATIONS: Occupations in Ohio where contact with arsenic was indicated are listed as follows:

Batch gatherers (glass factories)
Chemical operators (chemicals)
Chemists (explosives, ammunition, fireworks)
Color mixers (glass factories)
Cone fillers (explosives, ammunition, fireworks)
Cullet men (glass factories)
Factory foremen (explosives, ammunition, fireworks)
Factory laborers (explosives, amtion, fireworks)
Finishers (explosives, ammunition, fireworks)
Fireworks makers (explosives, ammunition, fireworks)
Laborers (electric fixtures)
Mixers (explosives, ammunition, fireworks; glass factories)
Platers (metal furniture)
Pressmen (explosives, ammunition, fireworks)
Tank men (glass factories)
Tree sprayers (foods)
Weigh car operators (electric fixtures)

Occupations which offer contact with arsenic and its compounds but not listed in the Ohio Survey are:*

Arsenic roasters
Artificial-flower makers
Artificial-leather makers
Bookbinders
Brass founders
Briquet makers
Bronzers
Calico printers
Candle makers
Carpet makers
Carroters (felt hats)
Chargers (zinc smelting)
Colored-paper workers
Compounders (rubber)
Copper founders
Copper smelters
Curriers (tanneries)
Decorators (potteries)
Electroplaters
Enamelers
Enamel makers
Farmers
Feather curers
Feather workers
Felt-hat makers
Ferrosilicon workers
Fur preparers
Galvanizers
Gardners
Glaze dippers (potteries)
Glaze mixers (potteries)
Gold refiners
Insecticide makers
Japan makers
Japanners
Lacquerers
Lacquer makers
Lead smelters

*Dublin, L.I., and Vane, R.J.: Occupation Hazards and Diagnostic Signs. U.S. Department of Labor, Bureau of Labor Statistics, Bulletin, 582:29-30, 1933.

Linoleum colorers
Lithographers
Mordanters
Painters
Paint makers
Paper glazers
Paper hangers
Paris-green workers
Pencil (colored) makers
Pitch workers
Pottery workers
Pressroom workers (rubber)
Printers
Pyrites burners
Refiners (metals)
Rubber workers
Sealing-wax makers
Sheep-dip makers
Shot makers
Soot packers
Sulfur burners
Sulfuric acid workers
Tannery workers
Taxidermists
Tinners
Toy makers
Velvet makers
Wallpaper printers
Wire drawers
Wood preservers
Zinc miners
Zinc refiners

MICRODETERMINATION OF ARSENIC.

Alfred E. How. Ind. Eng. Chem., Anal. Ed. 10, 226-32 (1938).

A modified Gutzeit procedure capable of detg. as little as 0.1 y As with a probable error of 5% and sensitive to 0.01 y As is described. The AsH_3 is evolved in a generator of new design and is absorbed on a string impregnated with $Hg-Cl_2$ from an alc. soln. and held in a capillary tube. The color is developed with 2% $AgNO_3$ in ammoniacal soln. and the length of the stain is measured to 0.01 cm. Study of a large no. of reagents resulted in the selection of (1) digestion mixt.: 60% $HClO_4$, concd. HNO_3 and concd. H_2SO_4 in the ratio 1:1:4; (2) alloy: Zn, Sn, Pb and Fe in the proportion (by wt.) 99.5:0.5:0.01: approx. 0.0028; (3) $NaHSO_3$ for reducing quinquevalent As; (4) $Pb(OAc)_2$ for removing H_2S. Many details of app., reagents and procedure, reagents and procedure, selected after esptl. evaluation and comparison, are given. Sixty-three references.

ACUTE ARSENIC POISONING.

W.A. Porter. Virginia Med. Monthly, vol. 66, pp. 148-151 (March 1939).

Abstracted in J. of Ind. Hygiene, vol. 21, no. 7, p. 169 (abstract section) Sept. 1939.

Besides the usual treatment of arsenic poisoning Porter used 800 international units of vitamin B_1 daily. The salient feature in the patients receiving the vitamin was the absence of diarrhea. It is his belief that the use of vitamin B_1 has a definite place in the treatment of toxic neuritis from metal poisoning. Prompt improvement of neuritic pains occurred after the vitamin B_1 had been administered for only a few days.--J.A.M.A.

EARLY DIAGNOSIS OF OCCUPATIONAL ARSENIC POISONING.

G. Straube. Deutsch. med. Wchnschr., vol. 65, pp. 334-335 (1939).

Abstracted in J. of Ind. Hygiene, vol. 21, no. 6, p. 141 (abstract section) June 1939.

In examining vineyard workers the author has attempted to distinguish arsenic absorption from arsenic poisoning. He believes those cases showing only hyperkeratoses may be considered as suffering from arsenic absorption, and that the presence of horny pearls is characteristic. These can be discovered in an early stage by the use of filtered ultraviolet light.--L. Teleky.

RESEARCH ON THE SIGNIFICANCE OF ALCOHOLISM IN THE DEVELOPMENT OF ARSENIC POISONING.

E. Zimmermann and E. Remy. Arch. f. Gewerbepathol., vol. 7, pp. 486-496 (1936).

Abstracted in J. of Ind. Hygiene, vol. 19, no. 5, p. 110 (abstract section) May 1937.

It has frequently been asserted that in several industrial poisonings a chronic alcoholic is much more endangered than workers who drink only

a little or not at all. The authors have fed hogs arsenic in the form of Fowler's solution in quantities 10 times the fatal dose for man. Besides the arsenic some animals were given alcohol in the form of beer to which they readily became accustomed so that even 500-550 cc. of alcohol produced no effect. At autopsy no special objective findings were seen. Chemical examination of the organs showed that in the animals given alcohol there was more arsenic than in the organs of the others--in the muscles, three times as much, three times as much in the liver, and in one other there was 12 times as much in the liver.--L. Teleky.

CASES OF ARSENIC POISONING.

P. Cristol, J. Fourcade, J. Ravoire and C. Bezenech. Rev. Hyg. et Med. prev., vol. 61, pp. 363-372 (May 1939).

Abstracted in J. of Ind. Hygiene, vol. 21, no. 8, p. 191 (abstract section) Oct. 1939.

Series of cases. One of severe dermatitis over almost the entire body. Analysis of the skin scales gave a value of 150 mg./kg. A second man lived near a metallurgical plant that gave off quantities of arsenious anhydride. Since 1932 he suffered pain, sometimes severe, and edema of the genitals, which gradually spread to his arms and legs. It was severe enough to make him cease work. Hair, pubic hair and nails gave As in amounts ranging from 64 to 280 mg./kg. Two additional cases of polyneuritis of agricultural and vineyard workers are described.--Helen Lawson.

CASE OF INDUSTRIAL ARSENIC CARCINOMA.

S. Miyaji. Zntrlbl. f. Chir., vol. 62, pp. 2063-2066 (1935).

Abstracted in J. of Ind. Hygiene, vol. 18, no. 8, p. 137 (abstract section) Oct. 1936.

A 54 year old man developed arsenical keratoses on both feet and an arsenical epidermoid carcinoma on the sole and dorsum of the left foot after 16 years of labor in an arsenic plant.

The lesions developed in spite of special precautions taken against inhalation or absorption through the skin of arsenic compounds. The left foot was partially amputated and the inguinal nodes were removed. The end-result is not stated. The article is illustrated with photographs.--Am. J. Cancer.

OCCUPATIONAL DERMATOSES DUE TO ARSENIC.

D. Lapysheff. Abstr. as follows from Sovet. Vest. Dermat., Feb., 1931, vol. 9, p. 126, in Arch. Dermat. and Syph., Dec., 1931, vol. 24, pp. 1089-1090.

Abstracted in J. of Ind. Hygiene, vol. 14, no. 4, p. 92 (abstract section) April 1932.

The author describes occupational skin diseases in workers who prepare a powder used for the destruction of corn blight which contains 75 per cent talcum and 25 per cent of one of the following three arsenic compounds: arsenious acid (As_2O_3), calcium arsenite ($Ca_3(As_2O_3)_2$) and Schweinfurtgreen, a double salt of copper and arsenic ($CuAsO_4(Cu_2H_2O_2)_2$). Of twenty-five workers examined twenty-one had skin affections in the form of acute and

subacute eczema, located mainly in the folds of the genitals, axillae, and antecubitals but also on the face and neck. Most of them showed also signs of secondary pyodermia in the form of folliculitis and perifolliculitis. Almost all had hyperemia and erosions of the mucosa of the nose, mouth, and pharynx. The majority showed signs of general poisoning (headache, malaise, and anorexia). In all of them a history was obtained of prodromal sensations antedating the eruption from seven to twelve days. The course was favorable; the patients became well soon after leaving their occupation. The author sees the main cause of these conditions in the technically bad arrangement of the factory and the insufficient sanitary and hygienic measures.

CHRONIC ARSENIC POISONING FROM WINE-INSECTICIDES.
Theodor Schondorf. Z. klin. Med. 133, 713-21 (1938).

A description of 12 cases of chronic As poisoning in workers in the wine industry in the palatinate of the Rhine, characterized by keratosis, melanosis, liver damage, gastric hypoacidity and other symptoms. Four of the patients were not actually in contact with the insecticide.

ACUTE ARSENIC POISONING FROM POTATO BUG INSECTICIDE.
H. Symanski. Samm. v. Vergiftsf. (gutachten), vol. 10, pp. 1-8 (Feb. 1939).
Abstracted in J. of Ind Hygiene, vol. 21, no. 7, p. 169 (abstract section) Sept. 1939.

A man sprayed an arsenic insecticide for about 3 hrs. That night he vomited and had diarrhea; the next morning he was unable to cut bread as usual for his lunch, but could do other work. The arm became weaker and lacked feeling; he could lift it from the shoulder but could not bend it. When he was examined about 2 wks. later the arm was colder than the left, weak, and somewhat reduced in circumference. Sensibility was decreased. These symptoms were somewhat less pronounced 3 mos. later. The findings are given in detail.--Helen Lawson.

ATMOSPHERIC POLLUTION WITH ARSENICAL DUST.
G. Sowden. Abstr. as follows from Jour. State Med., 1927, vol. 35, pp. 668-670, in Bull. Hyg., April 1928, vol. 3, pp. 286-287.
Abstracted in J. of Ind. Hygiene, vol. 10, no. 8, pp. 187-188 (abstract section) Oct. 1928.

This article records the discovery of arsenic (as As_2O_3) up to 500 parts per million in dust from the flue, and 150 parts per million in dust from the rain water gutter, of a factory--the source being the ordinary coal used as fuel.

Arsenic to the extent of 0.01 grain (the amount which should not be exceeded by, e.g., that in 1 pound of apples) is thus contained in 20 grains of such flue dust, which might easily be inhaled by a man sweeping such flues. As, however, the minimum lethal dose of 2 grains would only be ingested in $\frac{1}{2}$ pound of such dust, a considerable amount of chronic arsenic poisoning might occur without a death to lead to detection of the cause.

A HITHERTO UNSUSPECTED SOURCE OF ARSENIC IN HUMAN ENVIRONMENT.

R.E. Remington. Jour. Am. Chem. Soc., June 1927, vol. 49, pp. 1410-1416.

Abstracted in J. of Ind. Hygiene, vol. 9, no. 10, p. 183 (abstract section) Oct. 1927.

This article is summarized as follows:

Samples of American smoking and plug tobacco have been examined for arsenic, and found to contain from six to thirty parts per million, or from 0.05 to 0.27 grain of arsenic trioxide per pound.

Approximately half of the arsenic in pipe tobacco is evolved in the smoke, and about half of that in plug tobacco is soluble in water.

Amounts of arsenic reported are much in excess of the maximum permitted by state and Federal authorities in foods, and of the amounts normally present in plants and animals.--P.D.

ARSENIC IN TOBACCO SMOKE.

C.R. Gross and O.A. Nelson. Am. Jour. Pub. Health, Jan., 1934, vol. 24, pp. 36-42.

Abstracted in J. of Ind. Hygiene, vol. 16, no. 4, p. 81 (abstract section) July 1934.

An apparatus is described for use in determining volatile arsenic and other substances evolved during the smoking of cigars, cigarettes, and pipe tobacco.

Analyses of popular domestic brands of cigars, cigarettes, and smoking tobaccos show a range from 8.3 to 50.0 parts per million of arsenious oxide as compared with a range of 7 to 38.5 parts per million found by Remington in domestic smoking and chewing tobaccos.

On the basis of average figures, the arsenic inhaled in smoking 1.35 lb. of cigars, 0.57 lb. of cigarettes or 0.16 lb. (2.6 oz.) of pipe tobacco is calculated to be equivalent to that present in 1 lb. of food containing 1.43 parts per million of arsenious oxide which is the maximum permitted by law for food products.

The proportion of total arsenic volatilized during smoking ranges for cigars from 15.1 to 34.7 per cent, for cigarettes from 32.2 to 41.3 per cent, and for pipe tobaccos from 26.1 to 32.8 per cent. Remington reported about 50 per cent volatile in similar tests on pipe tobaccos.--Author's summary.

TOXICOLOGY OF PHENYLDICHLORARSINE. II. RESPONSE OF MAN TO PDA-OIL MIXTURES.

R.R. Sayers and H.C. Dudley. U.S. Pub. Health Repts., vol. 53, pp. 1292-1301 (July 29, 1938).

Abstracted in J. of Ind. Hygiene, vol. 20, no. 9, pp. 196-197 (abstract section) Nov. 1938.

This is the second paper on the toxicology of phenyldichlorarsine (PDA), the first being a report of animal experiments. Petroleum distillates containing 1% PDA are efficient wood preservatives. There is individual susceptibility to PDA-oil mixture, as demonstrated by skin tests, causing marked initial vesiculation is some, no reaction and delayed reaction in others. A ferric hydroxide-petroleum jelly will prevent serious skin lesions when spread over the exposed skin area.

Impregnated wood free from surface oil caused no reaction when in contact with the skin for as long as 1 hour. Steaming after impregnation removes surface oils. Thus, the hazard would appear to be controlled if in the impregnation process, which should be carried out in an entirely closed process, no excess oil remained on the surface of the impregnated timber. --George M. Reece.

OCCUPATIONAL POISONING BY ARSENOBENZENES.

Slosse. Abstracted as follows from Bull. de l' Acad. roy. de Belgique, 1921, vol. 1, p. 416-428, by. M. Heidelberger in Chem. Abstr., March 10, 1922, 16, No. 5, 754-755.

Abstracted in J. of Ind. Hygiene, vol. 4, no. 5, p. 66 (abstract section) Sept. 1922.

"Intoxications with varying manifestations and of varying intensity were observed among the physicians and attendants administering As antisyphilitics. Tests were made by Strzyzowski's modification (Oesterr. chem. Ztg., 1904, No. 4) of the Marsh test. Arsenic was found in the blood in four out of six cases, while normal controls showed none. In 1-g. samples of cleaned and degreased hair all the subjects showed As, which can not be detected in 1 g. of hair from normal persons. In nails 0.05- 0.3 g. was sufficient to give a positive test. Hippuric (A) and glucuronic (B) acids in the urine were higher than normal, which is taken to indicate the presence of the benzene part of the arsenobenzenes in the organism. Tests with respired air showed that the substances were not absorbed in this way, but a normal patient in whose socks arsenobenzene was placed showed a rapidly increasing amount of urinary A and B and gave a positive blood test, showing that absorption took place through the skin."

CAUTION WITH ARSINE.

H. Durr. Runschau deut. Tech. 18, no. 50, 9 (1938).

AsH_3 in concn. of 0.05 mg. per 1. of air is fatal within 30 min. to 1 hr. This gas occurs in metal works and chem. factories. Filter paper soaked in $HgCl_3$ and hung in the working rooms is recommended as an indicator for AsH_3.

ARSINE POISONING, REPORT OF TWO CASES.

K.D. Smith and T.E. Rardin. Ohio State Med. J., vol. 35, pp. 157-159 (Feb. 1939).

Abstracted in J. of Ind. Hygiene, vol. 21, no. 5, p. 117 (abstract section) May 1939.

Arsine and arsine poisoning are discussed and a description is given of the processes where the poisoning may be encountered. As is an impurity

in many metals and AsH_3, the most poisonous of the inorganic As compounds, is given off whenever these metals are acted upon by acids. The stripping of galvanizing with HCl is a common procedure and it was in this manner that the cases reported by the authors occurred. Complete descriptions are given of the two cases. The authors stress the importance of occupational histories, in addition to past illnesses and family history, as an aid in the diagnosis of vague cases.--W.H. Buck.

OCCUPATIONAL ARSINE POISONING OF A PHYSICIAN.

E.W. Baader. Samm. v. Vergiftungs., vol. 6, pp. 239-240 (1935).

Abstracted in J. of Ind. Hygiene, vol. 18, no. 4, p. 51 (abstract section) April 1936.

A doctor of internal medicine wished, for purposes of instruction, to demonstrate the determination of arsine by the Marsh method; he put zinc, sulfuric acid, and a small amount of arsenic in an Erlenmeyer flask which he left open in front of him for 5 minutes. Nausea developed very soon, and after $1\frac{1}{2}$ hrs. he could no longer stand; there was blood in the urine and he was jaundiced. For 6 weeks he was unable to work. The author suggests that arsine in its pure form does not always have the characteristic garlic odor.--L. Teleky.

THE "HAFF DISEASE".

Lancet, March 14, 1925, vol. 1, p. 566.

Abstracted in J. of Ind. Hygiene, vol. 7, no. 7, p. 122 (abstract section) July 1925.

Fishermen of the Kurisches Haff in Eastern Prussia experienced severe muscular pains, scanty urine with albuminuria and casts; 600 cases occurred with three deaths. The agent of the disease was finally discovered to be arseniuretted hydrogen. Certain cellulose factories had been using Rio Tinto pyrites containing arsenic; the effluent gained access to the bay, and 28 mg. of arsenic was found per liter of water; there the marine algae took up the poison and generated arseniuretted hydrogen. Arsenic was found in the blood, urine, and various organs of men and animals who died from the disease. Cyprian pyrites has been substituted and the disease has disappeared.--E.L.C.

CHRONIC ARSINE POISONING.

L. Schwarz. Samm. v. Vergift., vol. 9, pp. I-8 (1938).

Abstracted in J. of Ind. Hygiene, vol. 20, no. 7, p. 148 (abstract section) Sept. 1938.

A man employed as vat welder and galvanizer, fell ill with symptoms that increased gradually over a year's time; the examination showed paleness, slight yellowing of the sclera, cyanosis, r. b. c. 2.1 million, blood pigment 44%, tachycardia. The material used in welding and galvanizing was shown to contain more or less arsenic, and in some of the work processes, AsH_3 was found. The acetylene used contained AsH_3 and PH_3. The author therefore concluded, rightly, that a chronic AsH_3 poisoning was present. --L. Teleky.

CHRONIC ARSINE POISONING.

M. Kiese. Arch. f. exp. Pathol. u. Pharmakol., vol. 186, pp. 337-376 (1937).

Abstracted in J. of Ind. Hygiene, vol. 19, no. 10, pp. 221-222 (abstract section) Dec. 1937.

In industrial pathology the question of chronic arsine poisoning has not been clearly answered. The author has studied it experimentally by exposing mice, rats, guinea pigs, rabbits, cats and dogs for 3 to 5½ wks. for about 7 hrs. weekly to 4 to 50 gamma per L. air. A concentration of 4-12 gamma produced no effects in dogs, while 100 gamma given over a week produced subacute poisoning. At 36 gamma there was slight transitory general damage in the first few days and definite damage at 50 gamma in the course of the first week. Injuries concerned chiefly the blood, as in acute arsine poisoning; there was increased blood destruction--fall of erythrocyte count, and hemoglobin. Whereas at the beginning of the poisoning very active regeneration was seen, this frequently disappeared in the course of the exposure, resulting in a permanent anemia that in most of the animals reached a stationary condition. In the surviving animals, blood recovery took place more gradually than that of the general condition. Animals autopsied after the exposure showed a great deal of iron pigment in spleen, liver and bone marrow; damage to other organs was slight. The arsenic content of the organs was very unequal: spleen, liver, bone marrow and erythrocytes had the most. The amount in the hair after prolonged exposure was very high-up to 870 gamma %. The arsenic excretion remained about the same for several days when the intake was constant, but fell quickly at the end of the exposure and after 1-3 wks. again reached the physiological value. There must be a concentration range at which (as in uninterrupted arsine inhalation), the accumulation of poison and injury cannot reach its maximum. This range apparently lies at 20-25 gamma per L. One must assume that short inhalation of concentrations not fatal even if breathed without interruption will do less damage than uninterrupted exposure. A real habituation seems to develop.--L. Teleky.

POISONING BY ARSENIURETTED HYDROGEN AT THE ZINC SULFATE PLANT OF A LEAD WORKS.

Spangenberg and Kotzing. Zent. f. Gewerbehyg. u. Unfallverhutung, vol. 22, pp. 200-202 (1935).

Abstracted in J. of Ind. Hygiene, vol. 18, no. 8, pp. 127-128 (abstract section) Oct. 1936.

Three cases of poisoning by arsene are recorded, two of which were fatal. In the process which was used for the preparation of zinc sulfate there are three filtrations after dissolving the zinc in sulfuric acid; the first, to remove lead, the second, iron and arsenic, and the third, copper; in each case these substances were precipitated chemically--as no iron had been found present the management decided to omit the second filtration, and so a considerable amount of arsenic was left when the process of the third filtration was carried out. In this process neutralization is done by the addition of a calculated quantity of sulfuric acid and, owing to the presence of zinc, nascent hydrogen was produced which combined with the arsenic to form arsene. Contrary to instructions the acid had been poured direct into the vat instead of being passed through a long tube,

in consequence the men were exposed to any gases given off. One man engaged on this work went to the hospital for hematuria and was followed 3 days later by two other men. These two were seized with vomiting while at work, went home and were admitted to the hospital the next day. They both had bronze colored skin, pains in the back, hematuria, frequent vomiting, diarrhea and both developed complete anuria, one dying 7 days after admission and the other 8 days after. In both cases the number of r. b. c. was reduced to about a million and a half in 1 cmm.

As preventive measures it is recommended that the iron filtration never be omitted and that if no iron is present in the zinc used some be added to assist in the removal of any arsenic present, and that before the final process takes place the quantity of arsenic be estimated. Strips of mercury-bromide paper, which show by a yellow color the smallest trace of arsine, should be hung in the work rooms. For rescue work masks prepared with silver nitrate should be kept ready.--Bull. Hyg.

CASE OF LETHAL POISONING PROBABLY CAUSED BY ARSINE.

E. Laborde. Abstr. as follows from Ann. med. legale Criminol. police scient., 1927, vol. 7, pp. 651-654, in Chem. Abstr., March 20, 1928, vol. 22, p. 999.

Abstracted in J. of Ind. Hygiene, vol. 10, no. 9, p. 210 (abstract section) Nov. 1928.

Of two workmen who had cleaned sludge out of a tank which had contained sulphuric acid with 0.1 per cent arsenic, one had slight gastro-intestinal troubles and light bronchitis while the second died in forty-eight hours. Analysis of the viscera showed the presence of arsenic in quantities which, though small, were larger than the normal arsenic content of human viscera. From the postmortem examination and the circumstances of the case, Laborde concludes that death was due to arsine, probably evolved by action of the arsenic in the sulphuric acid on finely divided iron in the sludge.

FATAL ARSINE POISONING IN GALVANIZING.

F. Kunkele and H. Saar. Samm. v. Vergift., vol. 8, pp. 185-188 (1937).

Abstracted in J. of Ind. Hygiene, vol. 20, no. 2, p. 44 (abstract section) Feb. 1938.

In attempting to construct a galvanic zinc bath according to a secret process and carry out experiments in it, two workers fell ill and one died. Clinical and autopsy findings showed the cause to be arsine poisoning. Experiments with the chemicals used showed that the bath contained 1% As. When the zinc electrodes were put in, gas developed. The nascent H in the electric bath acted as a reducing agent on the arsenous compounds. In a great percentage of work experiments it has been shown that about 40 mgm. AsH_3 would be produced in 5 minutes.--L. Teleky.

ARSENIURETTED HYDROGEN POISONING DUE TO THE ACTION OF WATER ON METALLIC ARSENIDES.

R.R. Bomford, and D. Hunter. Lancet, December 31, 1932, pp. 1446-1449.

Abstracted in J. of Ind. Hygiene, vol. 15, no. 3, pp. 32-33 (abstract section) May 1933.

Aluminum is used in tin refining to remove from the tin, while in a molten state, copper, antimony, arsenic, and silver. Dross so formed is removed from the surface of the pot; it contains almuinum arsenide. In order to minimize dust, such dross was watered, whereupon arseniuretted hydrogen was generated. Two men working on the windward side were affected with slight attacks of haemolytic anaemia and jaundice. These cases might have passed unnoted, had not one case also suffered from an attack of lead colic. Arsenic was found in the urine in both cases. Clinical details are given of both cases, as well as references to other cases in past literature. The possibility of such a risk existing unsuspected in industry is pointed out, and methods for minimizing the risk are stated, including the use of birds in cages, hung near the work to act as indicators, since the gas affects them before it affects men.--E.L.C.

THE USE OF WHITE ARSENIC IN THE ENAMEL INDUSTRY.

M. Dechigi. Arch. f. Gewerbepathol., vol. 7, pp. 468-476 (1936).

Abstracted in J. of Ind. Hygiene, vol. 19, no. 5, p. 110 (abstract section) May 1937.

In the pottery industry to produce a fine white glaze, the cheaper arsenic trioxide is used in place of stannic oxide. The author shows that majolica tiles thus glazed when pulverized or broken up give off arsine in the presence of mold. He therefore urges protective measures against this source of domestic poisoning.--L. Teleky.

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